THE CLINICAL SIGNIFICANCE OF SYSTOLIC RETRACTION OF THE APICAL IMPULSE

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The clinical significance of systolic retraction of the apical impulse is still a controversial matter today. No general agreement exists as to whether systolic retraction may occur in health, some workers finding it to be the normal pattern in late systole (Eddlemann *et al.*, 1953), others that outward displacement persists throughout systole (Benchimol, Dimond, and Carson, 1961), and still others that either pattern may be present in health (Luisada and Magri, 1952). In constrictive pericarditis and adherent pericardium, on the other hand, systolic retraction of the impulse has long been an accepted physical sign (Broadbent, J. F. H., 1895; Broadbent, W., 1895; Dressler, 1937; McKusick, 1952; Goldberger, 1955). It has also been described in patients with great right heart enlargement and tricuspid incompetence (Dressler, 1937; Luisada and Magri, 1952; Goldberger, 1955; Salazar and Levine, 1962), though this association is probably less generally recognized.

In view of the continuing uncertainty about the meaning of systolic retraction of the cardiac impulse, we have attempted in this paper to reassess its clinical significance in health and disease.

SUBJECTS AND METHODS

The cardiac impulse was recorded in the impulse cardiogram: this is a record of what the hand feels and it measures total displacement. Details of the method have already been given elsewhere (Beilin and Mounsey, 1962; Deliyannis *et al.*, 1964). The impulse cardiograph is one of a number of different instruments that have been used in the studies of chest wall movements (Luisada and Magri, 1952; Eddlemann *et al.*, 1953; Benchimol *et al.*, 1961).

In this investigation, the areas of the chest wall selected for recording included the apex and the left and right parasternal areas. All records were made in held expiration. Impulse cardiograms were routinely recorded, with the patient lying on a Dunlopillo mattress, propped up in bed at an angle of about 45°.

The effect of posture on the impulse cardiogram was also studied in normal subjects, impulse cardiograms being recorded in three positions, prone, on the left side, and supine. In the latter case, the impulse recorder was mounted beneath the bed, and there was an opening in the mattress through which the impulse recording probe was introduced.

The 8 normal subjects, in whom the effect of posture was studied, were healthy men aged between 28 and 34. Antero-posterior chest radiographs of these subjects were taken in the supine position and also while they were lying on the left side, both being taken in held expiration.

There were 29 patients selected for study, of whom 4 had constrictive pericarditis, 22 had rheumatic heart disease, 2 had cardiomyopathy, and one had a left fibrothorax and systemic hypertension.

Of the patients with constrictive pericarditis, two had evidence on radiograph of a ring-like distribution of calcium in the pericardium of the annular constrictive type (Mounsey, 1959): one was successfully treated by
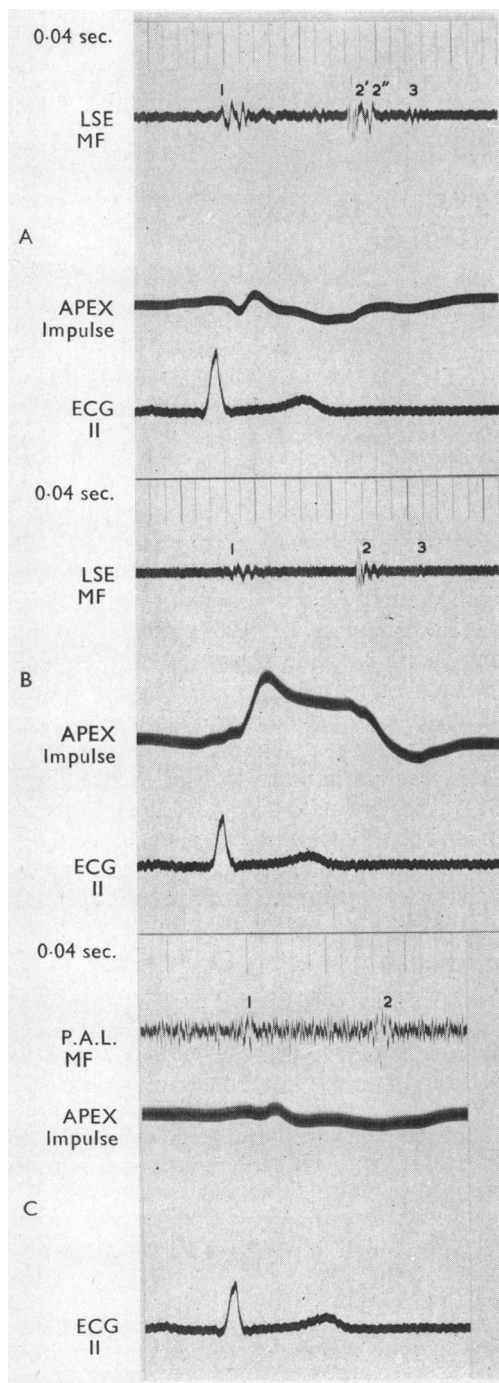
pericardectomy, and the other had milder symptoms and did not require operation. The other 2 patients were seen within a few months of the onset of tuberculous pericarditis, having already at that stage developed signs of constriction: in these cardiac catheterization and angiocardiography confirmed the diagnosis.

The 22 patients with rheumatic heart disease all had mitral valve disease: 16 had mitral stenosis (pure in 9 and with slight incompetence in 7) and 6 had dominant mitral incompetence (pure in 2 and with some stenosis in 4). In addition in 8 of these patients there was an aortic valve lesion, but in only one was this considered severe. Tricuspid incompetence was present in 13 patients as judged by a positive systolic wave in the jugular venous pulse, a pulsating liver, and a systolic murmur at the left sternal edge. Cardiac catheterization was carried out in 16 and selective left heart angiocardiography in 12 of these patients with a view to operation, but only 8 were selected for operative treatment.

Two patients in the series presented with heart failure of unknown ætiology with marked cardiomegaly and tricuspid incompetence. The underlying lesion was presumed to be cardiomyopathy. In one, after full investigation by cardiac catheterization, the possibility of constrictive pericarditis was excluded at thoracotomy, and in the other the diagnosis of cardiomyopathy was reached on clinical grounds alone.

Impulse cardiograms were recorded in every patient, and, where a change was noted clinically in the form of the impulse following successful treatment, the record was repeated. A simultaneous phonocardiogram and electrocardiogram were recorded as reference tracings. Additional information about cardiac movements underlying the impulses transmitted to the præcordium was obtained from cine radiological studies of the heart in 3 patients, using a simultaneous reference electrocardiogram.

General Note on Figures

Upward deflection in impulse cardiogram=outward movement of chest wall. Downward deflection=retraction of chest wall. LSE, left sternal edge; PA, pulmonary area; MA, mitral area; P.A.L. posterior axillary line; MF and HF, medium and high frequency phonocardiogram; 1, 2, and 3, first, second, and third heart sounds; 2', 2", split second sound; SM, systolic murmur; MDM, mid-diastolic murmur; OS, opening snap; EDS, early diastolic sound; ECG II, electrocardiogram lead II.
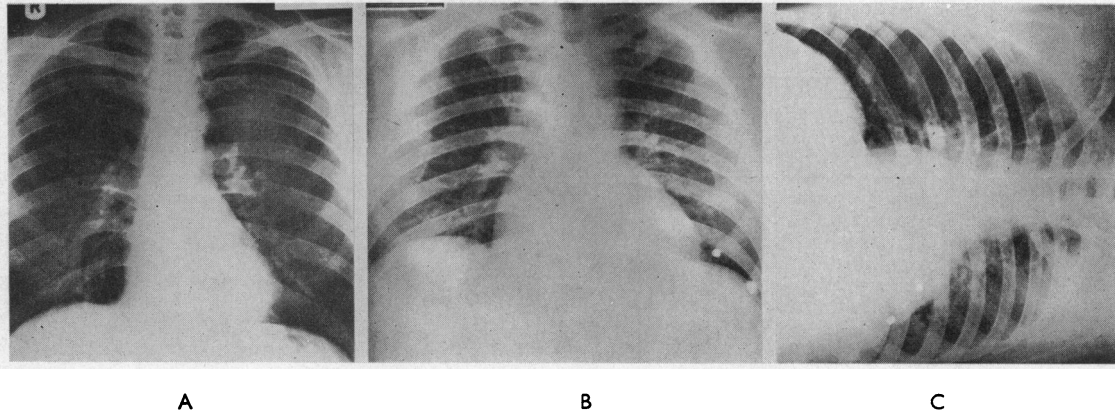

FIG. 1.—The apical impulse in health. (A) Subject supine. Two brief outward movements early in systole, followed by retraction. (B) Subject turned to left lateral decubitus position. Sustained outward beat lasting throughout systole. (C) Subject turned prone. Late systolic retraction is again present. (Upward deflection indicates outward movement. Downward deflection indicates retraction of chest wall.)

A   B   C

FIG. 2.—Chest radiograph of subject whose apical impulse is shown in FIG. 1. (A) 6 ft. A–P film in full inspiration with subject standing, showing normal cardiac shadow. (B) 4 ft. A–P film, in held expiration, with subject supine. (C) 4 ft. A–P film, in held expiration with subject lying on left side. There is compression of the left hemithorax, elevation of the left hemidiaphragm, and shift of the heart to the dependent side. The markers (white spots) indicate lateral displacement of the apex beat on turning on to left side. (Medial marker=apex beat supine; lateral marker=apex beat lying on left side.)

RESULTS

Apical Systolic Retraction in Health. In previous papers (Beilin and Mounsey, 1962; Deliyannis *et al.*, 1964; Gillam, Deliyannis, and Mounsey, 1964) the characteristics of the normal cardiac impulse in the impulse cardiogram have been described. In health the apical impulse imparts a short outward tapping sensation to the hand. It is caused by the front of the heart moving forward in early systole and striking the anterior chest wall which is moved outwards. During the latter part of systole the chest wall moves inwards again, as the heart retracts from it during late ejection (Fig. 1A).

The inward movement during the last third of systole was found to be an important characteristic of the healthy cardiac impulse, differentiating it clearly from the heave of ventricular hypertrophy that holds the chest wall outwards throughout systole (Beilin and Mounsey, 1962). This inward movement at the apex has been met in every normal subject examined in the supine position.

The extent of the inward movement in the impulse cardiogram varied. In most subjects the chest wall at the apex merely returned to its position before the onset of atrial and ventricular systole. In a few subjects, however, late systolic retraction was seen, the chest wall moving out again in the periods of diastolic rapid inflow and diastasis. The excursion of the systolic retraction was small, seldom exceeding 6 mm. in the cardiogram, which represents movement of as little as 0·1 mm. at the chest wall. Normal movements seldom exceed 0·2 mm. (Eddleman *et al.*, 1953).

The Effect of Posture. Since it was clear that late systolic retraction in health might well be to a certain extent dependent on the posture in which the recording was made, impulse cardiograms were recorded in 8 normal subjects, supine, in the left lateral decubitus position, and prone. It was found that posture did indeed considerably modify the form of the impulse, both clinically and as recorded in the impulse cardiogram. When turned from the supine to the left lateral position, 5 of the 8 subjects showed a change in the form of the impulse, from a short outward movement followed by slight retraction, to a sustained impulse resembling that seen in left ventricular hypertrophy in the supine position (Beilin and Mounsey, 1962) (Fig. 1B). That this was not a simple gravitational effect was shown by the fact that the same subjects lying prone (when gravity would tend to draw the heart against the anterior chest wall) once more showed retraction in late systole (Fig. 1C). The explanation of these changes was found on comparing chest x-rays taken supine and with the subject lying on his left side (Fig. 2). With the subject supine, the heart is centrally

TABLE I

CONSTRICTIVE PERICARDITIS

Patient No., sex, and age (yr.)	Diagnosis	Jugular venous pressure (cm. above S.A.)	Pericardial calcification	Left parasternal impulse	Apical impulse
1 F 54	Annular constrictive pericarditis	14	+	Retracting	Retracting
2 F 58	Annular constrictive pericarditis	8	+	Retracting	Retracting
3 M 30	Acute tuberculous pericarditis with constriction	14	0	No record	Retracting
4 M 23	Acute tuberculous pericarditis with early constriction	2	0	Normal	Normal

placed and supported by its mediastinal attachments (Fig. 2A and B). With the subject lying on his left side, the heart and mediastinum are displaced to the left, while the diaphragm rises; partial compression of the left lung resulting (Fig. 2C). Similar radiological findings have been noted by previous workers (Adams and Pillsbury, 1922; Webb, Forster, and Gilbert, 1921).

Since posture not only augments the amplitude of the cardiac impulse, but may also completely transform its character as a result of these changes of position of heart, mediastinum, and diaphragm, it seems essential to state clearly the position of the patient when the cardiac impulse is examined and recorded. In this clinical study, the supine position is used throughout, with the patient propped up at 45°, that being the normal position for examining the patient in the wards or in the outpatient department.

Apical Systolic Retraction in Constrictive Pericarditis. Patients with constrictive pericarditis may be broadly divided into two extreme groups: one having annular constriction, in which the main constriction occurs in a ring passing across the atrio-ventricular groove behind and over the pulmonary outflow tract in front (Mounsey, 1959); the other showing more generalized constriction, the whole pericardium acting like a rigid unyielding bag. Those with annular constriction usually have a rapid early arrested diastolic expansion of the heart on screening and kymography (McKusick, 1952), and on auscultation a loud early diastolic sound is heard. Such patients show systolic retraction of the apical impulse followed by a forcible palpable diastolic rapid inflow beat, often mistaken for the systolic impulse on casual examination. It is characteristic of these patients that retraction involves not only the apex but also the left parasternal area.

Two of the four patients studied (Table I) were of the annular constrictive group and showed marked systolic retraction followed by an abrupt rapid inflow beat in diastole (Fig. 3). One of these patients was treated by pericardectomy following which the impulse became more normal, the abrupt diastolic rapid inflow beat having disappeared (Fig. 4). The other two patients were seen earlier in their disease, when constriction was developing: one of these showed systolic retraction and diastolic outward movement of the præcordium as in the previous group: in the other, constriction was probably more generalized, the early diastolic sound was soft, and the cardiac impulse appeared normal both clinically and in the impulse cardiogram.

Apical Systolic Retraction in Mitral Valve Disease. The term "tapping" apical impulse of mitral stenosis really only applies to the palpable vibrations accompanying the first heart sound, at the moment of abrupt closing of the mitral valve. It does not describe the form of the displacement impulse throughout systole, which is what we are concerned with in this study.

In this investigation three types of apical impulse were noted in the presence of mitral valve disease, either pure or complicated by other valve lesions: a normal, a sustained, and a retracting impulse. The patients were divisible into 3 main groups.
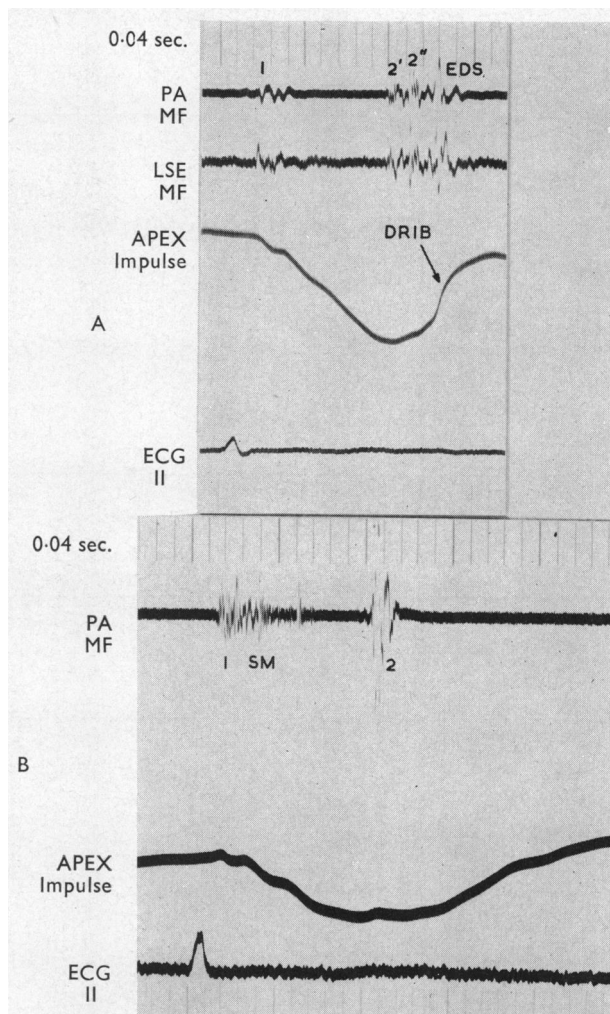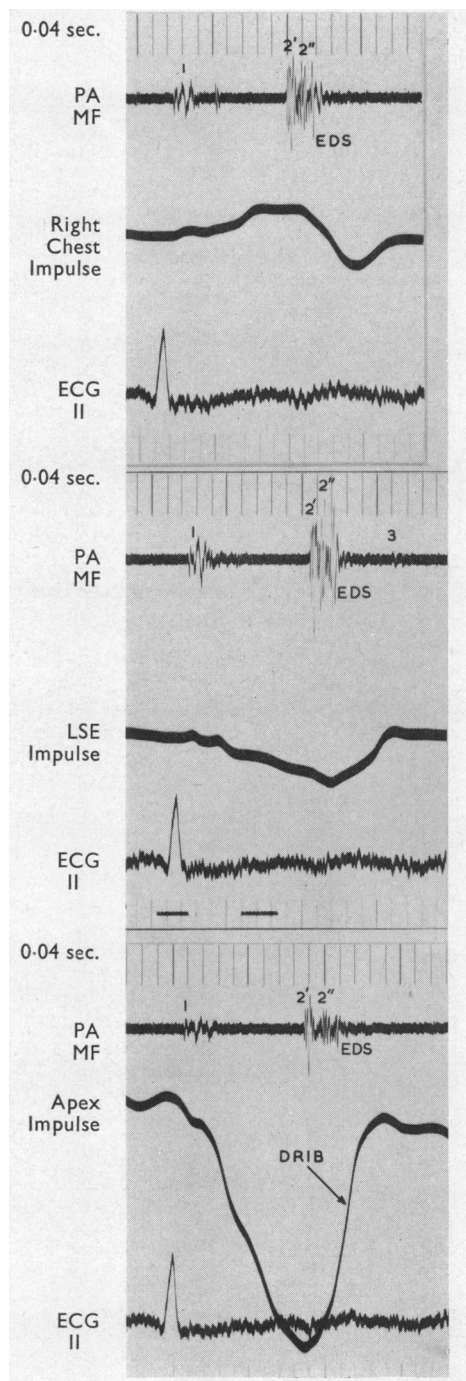
FIG. 4.—Constrictive pericarditis. (A) Before peri-cardectomy. Marked systolic retraction. Steep diastolic rapid inflow beat (DRIB) and early diastolic sound (EDS). (B) Two years after successful operation. Systolic retraction still present but steep diastolic rapid inflow beat and early diastolic sound have disappeared.

FIG. 3.—Impulse cardiogram in annular constrictive pericarditis. Marked systolic retraction at apex and lesser amount at left sternal edge. Systolic outward beat is present over the right chest. Steep diastolic rapid inflow beat (DRIB) at apex.

TABLE II
MITRAL STENOSIS

Patient No., sex, and age (yr.)			Diagnosis	X-ray	E.C.G.	JVP (cm. above S.A.)	PA pressure (mm. Hg)	Mean wedge pressure (mm. Hg)	Left para-sternal impulse	Apical impulse
1	F	55	MS	LA+ RV+	RV+	0	29/11	12	Normal	Normal
2	M	38	MS (MI) (AI)	LA+ RV+	RV+	0	28/24 (28)	15	Sustained	Normal
3	M	40	MS	LA+ RV+	RV+	0	48/24 (29)	19	Sustained	Normal overacting
4	F	42	MS	LA+ RV+	Digitalis effect	0	44/17 (22)	12	Sustained	Sustained
5	F	45	MS	LA+ RV+	Digitalis effect	0	32/20 (24)	18	Sustained	Sustained
6	F	67	MS	LA+ RV+ LV(+)	RV+	1	80/45 (50)	—	Sustained	Sustained
7	F	43	MS (AI)	LA+ RV+	Digitalis effect	3	RV 47/4	23	Sustained	Sustained

JVP, jugular venous pressure; PA, pulmonary artery; RV, right ventricle; LA, left atrium; LV, left ventricle; RA, right atrium; MS, mitral stenosis; MI, mitral incompetence; AI, aortic incompetence.

TABLE III
DOMINANT MITRAL STENOSIS WITH TRICUSPID INCOMPETENCE

Patient No., sex, and age (yr.)			Diagnosis	X-ray	E.C.G.	JVP (cm. above S.A.)	PA pressure (mm. Hg)	Mean wedge pressure (mm. Hg)	Left parasternal impulse	Apical impulse
1	F	63	MS (MI), TI	LA++ LV+ RA++ RV++	RV+	Angle of jaw	—	—	Sustained	Retracting
2	F	42	MS, (MI), (AS), (AI), TI	LA++ LV(+) RA++ RV++	RV+	8	—	—	Sustained	Retracting
3	M	36	MS, (MI), (AS), TI	LA++ RA aneurysmal RV++	RV+	10	—	—	No record	Retracting
4	M	45	MS, (MI), TI	LA+ RA+ RV++	RV+ LV (+)	13	68/41 (42)	20	Sustained	Retracting
5	F	15	MS, (TI)	LA++ RA+ RV++	RV+	5	51/27 (33)	25	Sustained	Retracting
6	M	55	MS, TI	LA+ RA(+) RV+	RBBB	10	50/25	17	Sustained	Retracting
7	F	70	MS, (MI), TI	LA+ LV(+) RA+ RV+	Digitalis effect	Angle of jaw	—	—	No record	Retracting
8	M	38	MS, (MI), TI	LA++ LV(+) RA+ RV++	RV+ LV(+)	8	57/34	18	Sustained	Retracting
9	F	40	MS, TI	LA++ RA aneurysmal RV++	RV+	10	33/16 (25)	19	Sustained	Retracting

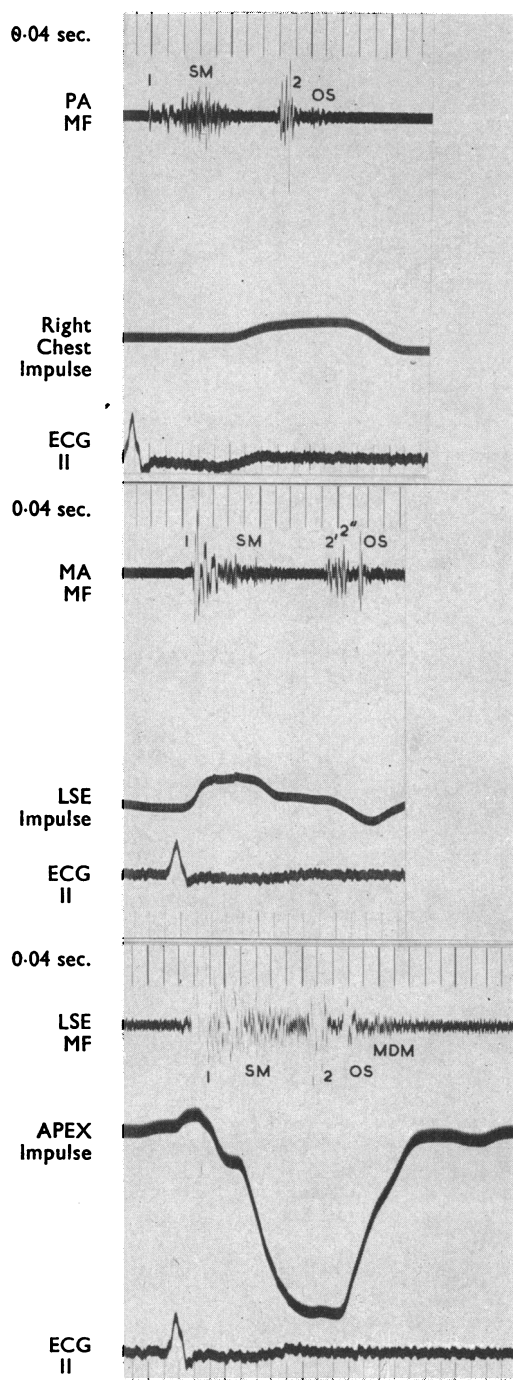TI, tricuspid incompetence; AS, aortic stenosis; RBBB, right bundle-branch block.

FIG. 5.—Rheumatic heart disease. Dominant mitral stenosis with free severe tricuspid incompetence. Deep systolic retraction at apex, small sustained systolic outward beat at left sternal edge, and small late systolic outward movement of right chest.

Group 1: 7 patients with dominant mitral stenosis and varying degrees of pulmonary hypertension (Table II). In 3 of these the apical impulse was normal, but in the remaining 4 the sustained left parasternal heave of right ventricular hypertrophy from pulmonary hypertension spread over to involve the apex (Deliyannis *et al.*, 1964; Gillam *et al.*, 1964).

Group 2: 9 patients with dominant mitral stenosis and in addition tricuspid incompetence of varying degree (Table III). In 4 of these patients (Cases 1, 2, 3, and 9) the tricuspid incompetence was free and associated with great enlargement of the right atrium, reaching aneurysmal proportions. In one patient (Case 5) tricuspid incompetence was slight and only present during an episode of right heart failure. In all 9 patients systolic retraction of the apical impulse was felt and recorded, with simultaneous outward sustained movement at the right sternal edge (Fig. 5). In the patient with slight tricuspid incompetence, apical systolic retraction disappeared after successful mitral valvotomy and relief of right heart failure (Fig. 6). In those with the largest right hearts and free tricuspid incompetence retraction was most marked. In one patient side-to-side rocking of the whole thorax was noted, of sufficient force to move the examination couch on which the patient lay. The apical systolic retraction contrasted with the sustained nature of the left parasternal impulse accompanying the right ventricular hypertrophy from pulmonary hypertension.

Cine films of the left and right cardiac borders in 3 of the patients with free tricuspid incompetence showed exaggerated retraction of the left heart border with simultaneous rightward bulging of the right atrial border (Fig. 7), reflecting the large regurgitant flow through the incompetent valve. Chest wall movements, therefore, closely mirrored the underlying heart wall movements.

Group 3: 6 patients with dominant mitral incompetence and in addition tricuspid incompetence of varying degree (Table IV). All these patients had combined radiological and electrocardiographic evidence of bi-ventricular hypertrophy. In all the apical impulse was sustained, reflecting underlying left ventricular hypertrophy (Gillam *et al.*, 1964) (Fig. 8). Apical systolic retraction was not seen in this group, in spite of free tricuspid incompetence in 5 of the 6 patients.
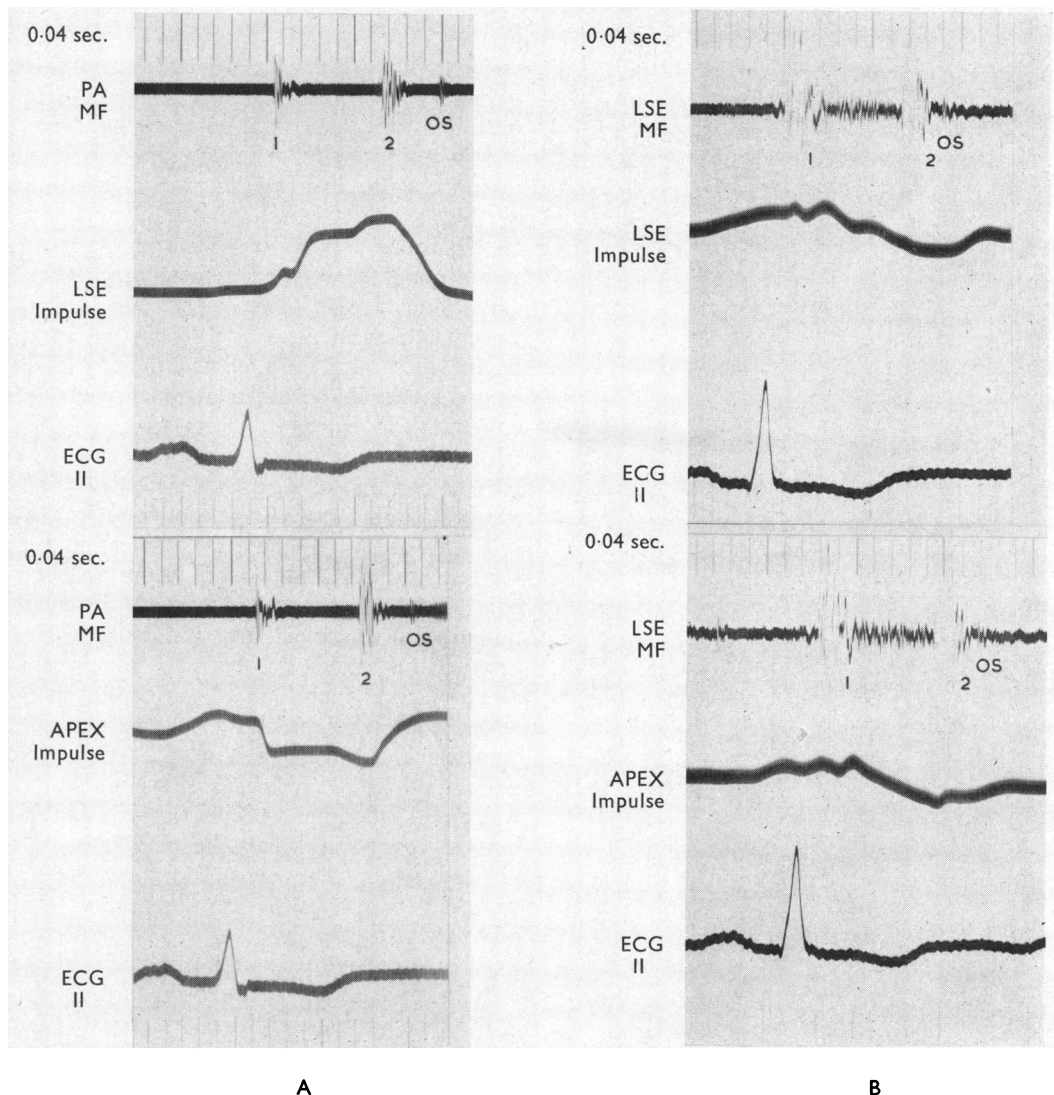
A   B

FIG. 6.—Pre- and post-operative impulse cardiograms in patient with mitral stenosis, pulmonary hypertension, mild tricuspid incompetence, and congestive heart failure. (A) Pre-operative systolic apical retraction and sustained impulse at left sternal edge. (B) 10 weeks after successful mitral valvotomy. Disappearance of tricuspid incompetence and congestive failure. Normal impulses at apex and left sternal edge.

Apical Systolic Retraction in Tricuspid Incompetence in Cardiomyopathy. Apical systolic retraction is not confined to tricuspid incompetence in rheumatic heart disease. Two patients, presenting with great right heart enlargement and chronic failure of unknown ætiology, were thought to have cardiomyopathy. Both had considerable tricuspid incompetence in association with chronic heart failure as judged by the positive systolic wave in the jugular venous pulse, a pulsating liver, and a systolic murmur at the left sternal edge. Both showed apical systolic retraction (Fig. 9).

Præcordial Systolic Retraction with Pleuro-pericardial Adhesions. One of our patients had diffuse fibrosis of the left hemi-thorax from an old tuberculous pleurisy with displacement of the heart to
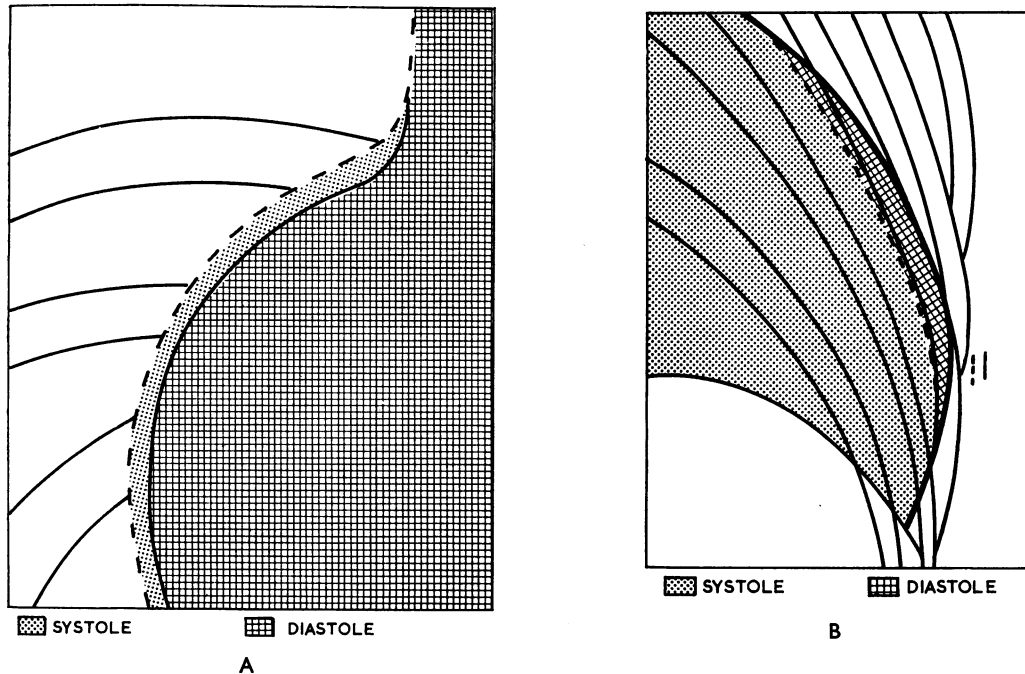
FIG. 7.—Tracings of cine film of the patient whose impulse cardiogram is shown in Fig. 5, showing systolic and diastolic movements of the right (A) and left (B) heart borders. (Film of left heart border in L.P.O. position with x-ray beam tangential to chest wall at apex beat.) Large rightward movement of both left and right heart borders in systole. Simultaneous retraction of apical impulse shown by movement of lead marker (right hand tracing).

TABLE IV

DOMINANT MITRAL INCOMPETENCE WITH TRICUSPID INCOMPETENCE

| Patient No., sex, and age (yr.)	Diagnosis	X-ray	E.C.G.	JVP (cm. above S.A.)	PA pressure (mm. Hg)	Mean wedge pressure (mm. Hg)	Left parasternal impulse	Apical impulse
1 F 40	MI, (AS), (AI), TI	LA++ LV+ RA+ RV+	LV+	4	—	—	Normal	Sustained
2 F 46	MI, TI	LA++ LV++ RA+ RV+	LV+ RV+	2	70/32 (42)	20	Sustained	Sustained
3 M 46	MI, (MS), AI, TI	LA+ LV+ RA+ RV+	LV+	8	85/48 (55)	—	Sustained	Sustained
4 F 37	MI, MS, AI, AS, (TI)	LA+ LV+ RA+ RV+	LV+	5	75/32 (55)	24	Bifid, sustained	Sustained
5 F 48	MI, (MS), (AI), TI	LA++ LV+ RA+ RV+	RV+	10	—	—	Sustained	Sustained
6 F 48	MI, (MS), (AI), TI	LA++ LV++ RA++ RV++	LV+	10	95/47 (55)	26	Sustained	Sustained
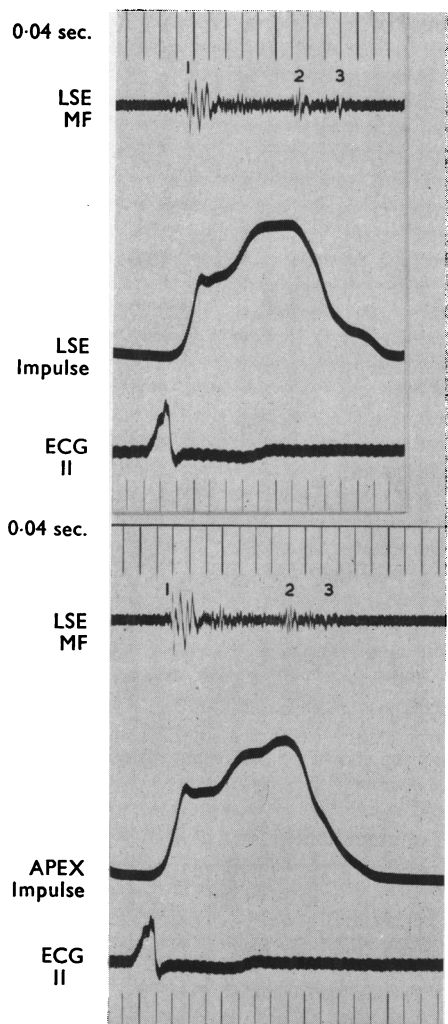
FIG. 8.—Rheumatic heart disease (MI, MS, AI, TI). Sustained systolic impulse at both apex and left sternal edge from combined ventricular hypertrophy.
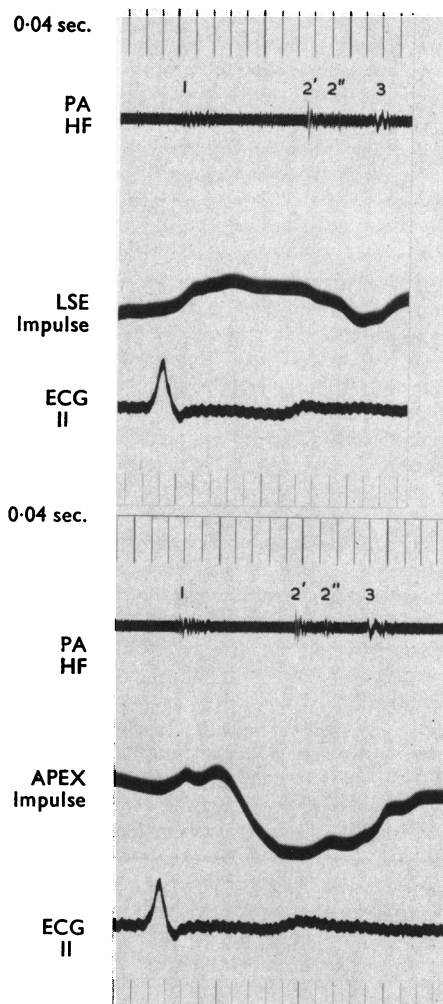
FIG. 9.—Cardiomyopathy with tricuspid incompetence· Systolic apical retraction and a sustained impulse at left sternal edge.

the left. The entire anterior præcordium showed striking systolic retraction, though in the mid-axillary line there was a sustained "apical" impulse due to left ventricular hypertrophy from systemic hypertension (Fig. 10).

DISCUSSION

Greater importance used to be attached to the physical sign of systolic retraction of the cardiac impulse as indicating adherence of the pericardium (Broadbent, J. F. H., 1895; Broadbent, W., 1895). When, however, it became apparent that the sign was also present in many cases of cardiomegaly without significant pericardial adhesions (Broadbent, J. F. H., 1895; Dressler, 1937; Wood et al., 1951) the original interest in the importance of the sign waned.

We have confirmed the fact that apical systolic retraction may occur both in constrictive pericarditis and in cardiomegaly without pericardial disease. In addition we have shown that the lesion

which appears to be responsible for systolic re-traction in the latter group is tricuspid incom-petence, in the absence of left ventricular hypertrophy. Distinction between a retracting cardiac impulse due to constrictive pericarditis and that due to tricuspid incompetence can usually be made on reviewing the whole præ-cordial impulse pattern. In constrictive peri-carditis, the whole of the left side of the præcordium retracts in systole (Wood *et al.*, 1951), only the right side of the chest showing sustained outward systolic movement. In tri-cuspid incompetence, on the other hand, right ventricular hypertrophy from right ventricular hypertension is usually present and this ac-counts for the sustained outward impulse at the left sternal edge, contrasting with simultaneous retraction at the apex.

Systolic retraction of the apical impulse is of clinical importance, first, because the sign is confined to three conditions, constrictive peri-carditis, tricuspid incompetence, and pleuro-pericardial adhesions, and secondly, because, if not recognized, it may be mistaken for the sus-tained impulse of ventricular hypertrophy, a systolic lift being confused with a diastolic lift on superficial examination. Extensive pleuro-pericardial adhesions must be remembered as a rare cause of systolic retraction of the apical impulse, but the absence of symptoms of heart disease, and in particular the normal jugular venous pressure, exclude associated constrictive pericarditis. Having noted the presence of apical retraction in the cardiac patient, we have shown how, on the impulse alone, distinction can be made between constrictive pericarditis and tri-cuspid incompetence.

The sign may also be of use in helping to assess complicated cases of polyvalvular rheu-matic heart disease. In the presence of tricuspid incompetence, it may be difficult to decide whether there is also associated mitral incompe-
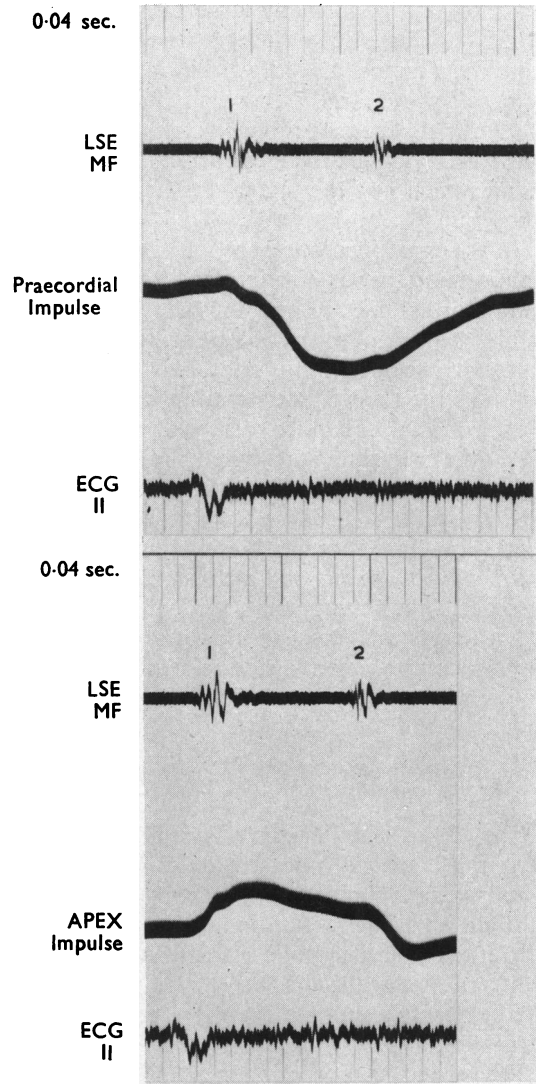

FIG. 10.—Left fibrothorax and hypertension. The apex beat was in the mid-axillary line and sustained throughout systole. Systolic retraction of the entire præcordium medial to the apex beat was present.

tence since the systolic murmur of tricuspid incompetence is often propagated to the apex. In our series of patients with tricuspid incompetence, apical systolic retraction was only seen with domi-nant mitral stenosis and never with dominant mitral incompetence.

The genesis of apical systolic retraction in constrictive pericarditis and tricuspid incompetence is probably fundamentally different, though both conditions have in common a raised atrial filling pressure. In annular constrictive pericarditis two different factors probably operate to produce apical systolic retraction, one in diastole and the other in systole. Tethering the heart over the atrio-ventricular groove and right ventricular outflow tract impedes the normal descent and ascent of these regions during systole and diastole. In diastole the right ventricle is seen at thoracotomy

to fill with an abnormally large outward movement of the free portion of the anterior ventricular wall.   The diastolic impulse pattern thus closely follows the underlying disturbed functional cardiac anatomy, a large steep outward movement coinciding with filling of the heart.   Its steepness reflects the rapidity and force with which the ventricle fills as a result of the high atrial pressure.   In systole, an additional factor probably operates in some cases, due to the presence of external pericardial adhesions.   These attach the pericardium to the anterior chest wall, which thus follows more closely the movements of the underlying heart, as was also the case in the patient with pleuro-pericardial adhesions.

In tricuspid incompetence, on the other hand, increased right ventricular stroke output accompanies simultaneous ejection of blood through the pulmonary and tricuspid valves (Korner and Shillingford, 1954).   Cine films show that this is partly accomplished by abnormally large inward systolic movement of the apical region of the heart, which is formed by the hypertrophied and dilated right ventricle (Deliyannis et al., 1964).   They also show simultaneous systolic expansion of the right atrium as the regurgitant stream pours into it from the right ventricle.   The systolic pattern in the impulse record, therefore, of apical retraction with simultaneous right chest expansion reproduces closely the underlying movements of the heart wall.   In early diastole the large outward movement of the apical impulse reflects the increased stroke input into the right ventricle and the raised right atrial pressure.   The large amplitude of the apical impulse in tricuspid incompetence probably also reflects the close coupling between heart and chest wall produced by the dilated right ventricle and atrium which are pressed against the overlying chest wall in diastole by the raised venous filling pressure.   The four patients with the most marked apical systolic retraction were those with the largest hearts and most marked tricuspid incompetence.   Another factor in patients with free tricuspid incompetence is a side-to-side rocking force imparted to the thorax as a whole, produced by the to-and-fro shunting of blood through the incompetent tricuspid valve in systole and diastole.

SUMMARY

The clinical significance of systolic retraction of the apical impulse has been investigated, using the impulse recorder described by Beilin and Mounsey (1962).   Eight healthy subjects and 29 patients were included in the study.

Slight apical retraction in later systole occurred in health, with the subject lying prone at 45°.

Marked apical systolic retraction occurs in two forms of heart disease.   In constrictive pericarditis of the localized annular type, widespread retraction of the whole præcordium, including the apex beat, was seen.   In the more generalized form of constrictive pericarditis, however, this sign was absent.

The second cardiac lesion in which apical systolic retraction occurred was tricuspid incompetence, most commonly due to rheumatic heart disease, but in two cases to cardiomyopathy.   Apical retraction did not occur in tricuspid incompetence, if the left ventricle was hypertrophied.   Retraction was most vigorous with free tricuspid incompetence and giant right heart enlargement: in an exceptional case side-to-side rocking of the whole thorax was noted.   In contradistinction to the widespread præcordial retraction of constrictive pericarditis, in tricuspid incompetence only the apical area retracted.

A rare cause of præcordial systolic retraction is pleuropericardial adhesions, in the absence of heart disease.

The genesis of apical systolic retraction is discussed.

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